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CHAPTER 11

AMNESIA

Alan Baddeley

We all have memory lapses, some more embarrassing than others. On one occasion, I agreed to appear on a live radio phone-in program, the *Jimmy Mack Show* from Glasgow. As I lived in Cambridge at the time, it was agreed that I would participate from the local radio station. That morning I was reading the newspaper before checking my diary and setting off for work, when I glanced at the TV and radio section, prompting the awful realization that I should at that moment be telling the world about the wonders of memory. I leapt on my bike and arrived just before the end of the program, sheepishly muttering about the terrible traffic in Cambridge, to be asked by the host if I could give the listeners a few hints on how to improve their memory!

So we all have bad memories, but what is it like to have a genuine memory problem—not the devastatingly dense amnesia experienced by Clive Wearing and described in Chapter 1, but the much more common level of memory deficit that accompanies many conditions including stroke, Alzheimer's disease, and traumatic brain injury? A very good account of the problems associated with memory deficit is given by Malcolm Meltzer, a clinical psychologist who experienced memory problems following a heart attack that led to anoxia (Meltzer, 1983).

Meltzer emerged from a 6-week coma knowing who he was and recognizing his family, but thinking he was 33 years old whereas in fact he was 44. On returning home, he could not remember where things were kept and, unlike a pure amnesic patient, also had problems in remembering skills such as how to set an alarm clock, when bills should be paid, where was a good place to go for a vacation, and how one might get there. He also had problems with his working memory:

> *Organization of thinking was hampered ... I had trouble keeping the facts in mind, which made it difficult to organize them ... comparing things along a number of variables is difficult to do when you cannot retain the variables.* (Meltzer, 1983, p. 4)

Meltzer found it hard work to watch films or TV because of the difficulty in remembering the plot or, in the case of sports, which team was which and which was ahead. He tended to find spatial orientation difficult and even walks in a familiar neighborhood were liable to result in his getting lost. A particular problem was the impact of his amnesia on his capacity to interact with people:

> *Having conversations could become a trial. Often in talking with people I was acquainted with, I had trouble remembering their names or whether they were married, or what our relationship had been in the past. I worried about asking where someone's wife is and finding out that I had been at her funeral two years before.*

> *Often if I didn't have a chance to say immediately what came to mind, it would be forgotten and the conversation would move to another topic. Then there was little for me to talk about. I couldn't remember much about current events or things I read in the paper or saw on TV. Even juicy tit-bits of gossip might be forgotten. So in order to have something to say, I tended to talk about myself and my "condition." My conversation became rather boring.* (Meltzer, 1983)

Eventually, with considerable perseverance, Meltzer recovered sufficiently to return to work, and of course to write a paper, providing for carers and therapists a very clear insight into the problems that result from memory deficit.

STUDYING AMNESIA

Unlike much research in cognitive psychology, the study of amnesia depends critically on clinical psychologists who are interacting with patients for practical and professional reasons. The clinical priorities vary from one center to another, with investigators from different backgrounds tending to emphasize different approaches. In the case of the Montreal Neurological Institute, as Brenda Milner's (1966) study of HM the classic case of impaired episodic LTM described in Chapter 2 demonstrates, there was a need for the neuropsychologist to identify the function of different brain areas so as to guide the neurosurgeon and minimize any unwanted side effects. Milner's approach therefore tended to stress anatomical localization, and be less concerned with the development of theory in cognitive psychology.

A second important pioneer in this area was Elizabeth Warrington, who works at the National Hospital for Nervous Diseases in London. This is the principal UK center for neurology, and often receives patients who show an unusual pattern of symptoms, presenting a challenge to diagnosis. Elizabeth was interested in linking the nature and location of their brain dysfunction with the development of a better functional understanding of the cognitive deficits shown by patients, and in this connection often chose to collaborate with mainstream cognitive psychologists.

A similar collaboration occurred in a third main research group concerned with amnesia, located at The Boston Veterans Administration Hospital, where a clinical neuropsychologist, Nelson Butters, worked with a cognitive psychologist, Laird Cermak. A major problem confronting the hospital was that of treating service personnel who had developed alcoholism, which in many cases resulted in Korsakoff syndrome, the principal source of amnesic patients for this group. Korsakoff syndrome is an alcohol-related problem in metabolizing the vitamin thiamine, which in turn leads to brain damage in areas such as the hippocampus and diencephalon, which are essential for episodic memory.

The difference between the practical demands facing these three groups can lead to apparent scientific discrepancies, potentially resulting in controversy. In fact, all three approaches are necessary but serve different functions. From the viewpoint of a cognitive psychologist, the most informative studies are those in which the deficit concerned is theoretically important and pure, regardless of whether it results from surgery, a stroke, or alcoholism. From a neurosurgical viewpoint, however, the link between function and anatomy is the crucial issue, with the anatomical localization of the patient's brain lesions being more important than the implications of the deficit for understanding normal cognitive function. Finally, if one wishes to understand a particular disease, be it Korsakoff syndrome, Alzheimer's disease, or schizophrenia, then it is essential to study patients with that disease, even though the nature, the purity, and extent of both their anatomical and cognitive deficits are likely to vary from one patient to the next. Of course, it is possible to select patients with relatively pure cognitive deficits, but symptom complexity is an important feature of the disease, and hence should be studied rather than avoided. These additional complicating features can, however, potentially give rise to controversy when, for example, a disease-based study is used to draw strong theoretical conclusions, as we shall see below.

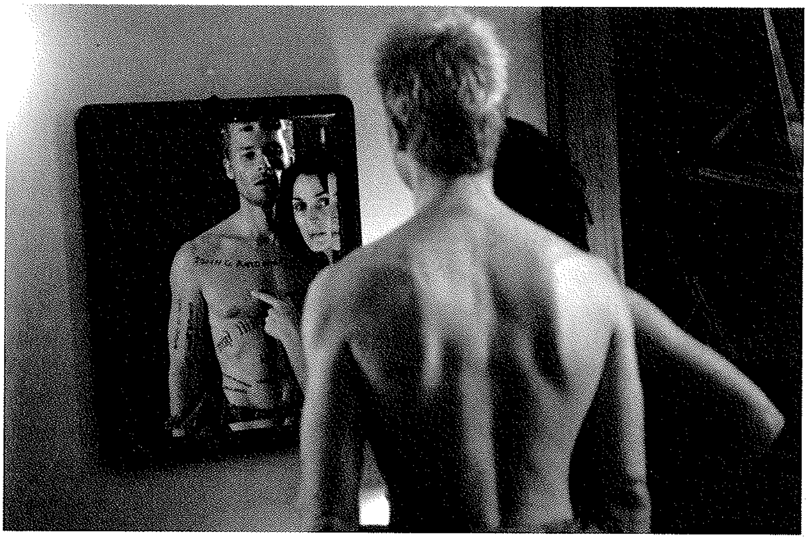
TERMINOLOGY

A crucial distinction is that between **anterograde amnesia** and **retrograde amnesia**. Anterograde amnesia refers to a problem in encoding, storing, or retrieving information that can be used in the *future*, hence the prefix *antero*. By contrast, retrograde amnesia refers to loss of access to events that happened in the *past*, typically before the onset of the disease. The densely amnesic patient HM, described in Chapter 2, is a classic case of anterograde amnesia because his capacity for new learning was greatly restricted but his ability to recall events from before his operation was relatively well preserved. In this respect, he can be contrasted with Boswell, a patient who was also densely amnesic and unable to acquire new episodic information, but who also was unable to retrieve factual knowledge from the past (Damasio, Eslinger, Damasio, Van Hoesen, & Cornell, 1985), and hence who suffered from both anterograde and retrograde amnesia. You might recall that Clive Wearing, who was described in Chapter 1, similarly showed dense anterograde amnesia together with retrograde amnesia reflected in his very patchy access to earlier memories.

Other types of amnesia include **post-traumatic amnesia** (PTA), a state that often follows a severe concussive head injury. A patient in PTA has difficulty forming new memories, a condition that tends to improve gradually over time (Levin & Hanten, 2002). Finally, **transient global amnesia** (TGA), is

KEY TERMS

Anterograde amnesia: A problem in encoding, storing, or retrieving information that can be used in the future.
Retrograde amnesia: A problem accessing events that happened in the past.
Post-traumatic amnesia (PTA): Patients have difficulty forming new memories. Often follows a severe concussive head injury and tends to improve with time.
Transient global amnesia (TGA): Apparently normal individuals suddenly develop severe problems in forming and retrieving new memories. The cause is unknown and the condition tends to resolve relatively rapidly.



The 2001 film *Memento* chronicles the story of Leonard, an ex-insurance investigator who can no longer build new memories, as he attempts to find the perpetrator of a violent attack that caused his post-traumatic anterograde amnesia and left his wife dead. The attack is the last event he can recall. © Corbis Sygma.

a condition in which an apparently normal individual suddenly develops severe problems in forming and retrieving new memories, a state that fortunately tends to resolve relatively rapidly. The cause of TGA, and indeed, the question of whether it has one or many causes, is still unclear (Goldenberg, 2002).

ANTEROGRADE AMNESIA

The amnesic syndrome

A classic case of the amnesic syndrome would have preserved intellect and language coupled with a dense impairment in the capacity for episodic learning, whether tested visually or verbally, and whether by recall or recognition. Whereas memory disruption might result from almost anything that interferes with the normal functioning of the brain, the pure but dense amnesia that constitutes the classic amnesic syndrome tends to be associated with a more limited range of different causes. These include bilateral damage to the temporal lobes and hippocampus, **alcoholic Korsakoff syndrome**, prolonged anoxia, and encephalitis resulting from brain infection. Any of these is likely to result in cognitive deficits that extend beyond amnesia, but all can on occasion result in a dense but pure deficit of episodic memory.

Elizabeth Warrington and I studied a group of patients varying in their etiology or source of disease but all having preserved intellect and grossly impaired episodic memory (Baddeley & Warrington, 1970). They were all densely amnesic, showing greatly impaired capacity for learning verbal or visual material, whether tested by recall or recognition. They had, however, preserved digit span and showed a normal recency effect in a task involving the free recall of 10 unrelated words, although performance on earlier items was markedly impaired (Figure 11.1).

Our patients also performed normally on the Brown–Peterson short-term forgetting task, not only in terms of their rate of forgetting but also in the point at which forgetting leveled off, well above baseline. This led to controversy, as Cermak, Butters, and Moreines (1974), testing a group of amnesic Korsakoff patients in Boston, observed impaired Peterson performance. They also found that their patients failed to show the normal release from proactive interference (see Chapter 2, p. 23) when the words to be remembered in the Peterson task were changed from one semantic category to another, for example from animals to flowers (Cermak & Moreines, 1976), whereas our patients showed the normal pattern, with performance improving dramatically on the first trial with the new category. The failure to find release in the Boston patients was attributed by Cermak

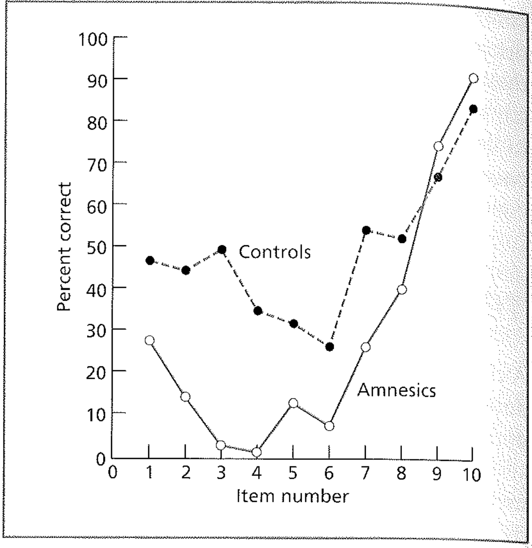


Figure 11.1 Immediate free recall in amnesic and control patients. Amnesic patients show preserved recency and impaired recall of earlier items. From Baddeley and Warrington (1970). Copyright © Elsevier. Reproduced with permission.

KEY TERM

Alcoholic Korsakoff syndrome: Patients have difficulty learning new information, although events from the past are recalled. There is a tendency to invent material to fill memory blanks. Most common cause is alcoholism, especially when this has resulted in a deficiency of vitamin B1.

and Butters to an absence of deep semantic coding, thus providing what appeared to be a neat explanation of amnesia in terms of the Craik and Lockhart levels of processing hypothesis (see Chapter 5, p. 99). They made a number of suggestions regarding possible methodological differences between our study and theirs that might explain the discrepancy between our results.

The issue was finally resolved when Cermak (1976) tested SS, a patient with a very pure amnesia resulting from a brain infection, who behaved like the London amnesic patients. Careful further investigation indicated that the Boston Korsakoff patients had a less pure memory deficit than was at first supposed. As is often—although not always—the case, the patients with Korsakoff syndrome showed subtle signs of damage to the frontal lobes resulting in impaired executive function. The Peterson task involves maintaining a memory representation at the same time as performing the demanding interpolated task of counting backwards. As such, it involves working memory and not simply verbal STM, and hence is sensitive to frontal lobe damage.

What can amnesic patients learn?

Despite their problem with new episodic learning, it has been known for many years that there are certain things that pure amnesic patients are able to learn. A classic anecdote from the Swiss neuropsychiatrist Claparède (1911) concerns an informal experiment he carried out on one occasion on the ward round in his clinic. Each day he would visit and shake hands with his patients. On one occasion, he secreted a pin in his hand when shaking hands with an amnesic woman. Next day she refused to shake hands but could not remember why. This is, of course, an instance of classical conditioning in which the handshake is associated with pain. Subsequent studies, under more controlled conditions, have shown that amnesic patients show normal classical conditioning. In one study (described in Chapter 4; see p. 79), a light was followed by a puff of air to the eye causing an automatic blink response (Warrington & Weiskrantz, 1978; Gabrieli, McGlinchey-Berroth, Carrillo, Gluck, Cermak, & Disterhoft, 1995). After a number of paired presentations of light and air puff, the light alone was able to evoke a blink, an instance of classical avoidance conditioning. However, confronted with the equipment and asked what it did, patients were quite unable to remember.

Priming

As noted in Chapter 4, Warrington and Weiskrantz (1968) found that, although their amnesic patients were grossly impaired in their capacity for recalling or recognizing items from a list of words or pictures, they performed as well as controls when asked to identify a visually degraded version of the relevant words or pictures. A similar result was obtained by Graf, Squire, and Mandler (1984), when they compared amnesic and control patients on two tasks. One was implicit and involved stem completion, a task in which a list of words (e.g. *METAL*) is presented and then tested by providing the initial letters (e.g. *ME* – – –) and asking participants to "guess" a word that would fit. The second task, cued recall, also involved presenting the word stem, but this time asking participants to *remember* what word had been presented. There was a clear difference between the two groups when given the explicit *remember* instruction, but not with the implicit stem completion task.

Skills

Corkin (1968) observed that HM showed normal performance on a task involving tracing a star observed through a mirror, a task that is initially frustratingly difficult but to which you rapidly adapt. Brooks and Baddeley (1976) tested amnesic patients on a pursuit rotor task in which you have to keep a stylus in contact with a moving target. Both patients and controls showed a similar rate of improvement with practice. As Figure 11.2 shows, novel perceptual tasks such as reading words

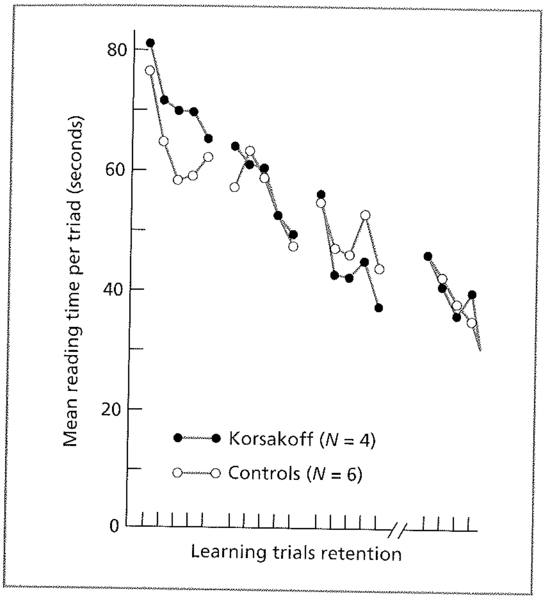


Figure 11.2 Acquisition of a mirror-reading skill across three daily sessions, and retention 3 months later. The amnesic Korsakoff patients learned and retained the skill as well as controls. Data from Cohen and Squire (1980).

printing in mirror-transformed script can also be learned by amnesic patients as readily as control participants (Cohen & Squire, 1980).

Controlling complex systems

Another form of implicit learning involves highly complex skills such as these involved in learning an artificial grammar or controlling a complex system simulating the operation of a sugar-producing factory (see Chapter 4, p. 85). Amnesic patients have been shown to be quite able to learn such skills (see Squire, Knowlton & Musen, 1993, for a review).

Hedonic adaptation

Johnson, Kim, and Risse (1985) were interested in the phenomenon whereby an unfamiliar experience, such as hearing a Korean melody for the first time, is often judged negatively, but then, with repeated presentations leads to increasingly positive judgments (see p. 81). They observed this effect in their amnesic patients, who nevertheless denied ever having heard such a melody before.

THEORIES OF AMNESIA

Although there is considerable agreement as to the phenomena associated with the amnesic syndrome, there is much less agreement on how to interpret them. One suggestion is that amnesic patients lack the capacity to consolidate memory traces (Milner, 1968, Squire & Alvarez, 1995; Cahill, Haier, & Alkire, 1996). This is essentially a physiological interpretation, although it clearly has implications for performance at the psychological level. A number of such explanations have been proposed at a more cognitive level, including:

Levels of processing

As noted earlier in this chapter, Cermak and colleagues, noting that their Korsakoff patients apparently failed to take advantage of semantic coding in a release from a proactive interference paradigm suggested that a failure to encode deeply might lie at the root of their memory problem. Cermak and Moreines (1976) attempted to test this hypothesis in a levels of processing study and found that their patients showed little advantage from the deep processing condition. However, a subsequent study indicated that this was due to a floor effect. The patients had shown so little learning in any condition that no differences could be detected. When the task was made easier, patients showed the standard levels of processing effect (Cermak, & Reale, 1978).

In a further investigation of this issue, Meudell, Mayes, and Neary (1980) used cartoons as their stimulus material. In the shallow encoding condition, participants looked for minor differences between two very similar drawings. In a second condition, they were asked to memorize the cartoons for later test, whereas in the deepest condition they were asked to describe the cartoons and rate them for humor. The patients with alcoholic Korsakoff syndrome performed more poorly overall, but as Figure 11.3 shows had retained a normal sense of humor and showed just as clear a depth-of-processing advantage as did controls.

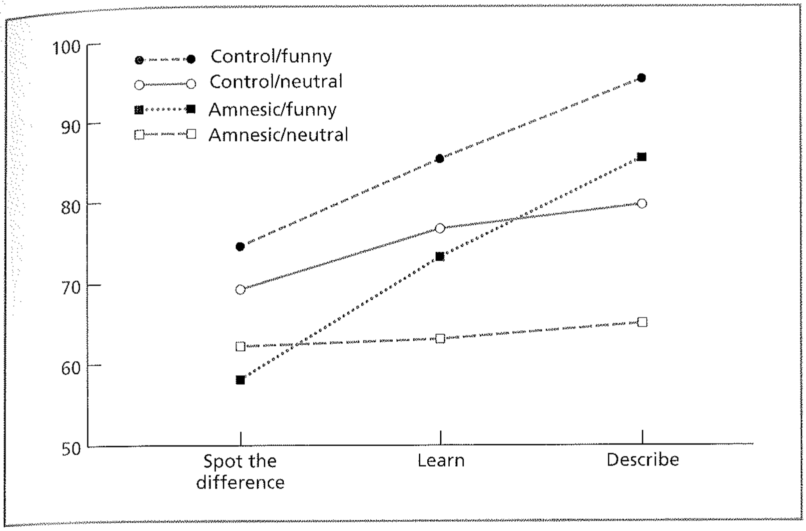


Figure 11.3 Effect of encoding instructions on subsequent recognition of humorous or neutral cartoons by amnesic patients and controls. Both groups are helped by humor and by deeper encoding instructions Adapted from Meudell et al. (1980).

Faster forgetting?

There are at least two versions of the consolidation hypothesis. The first assumes that long-term traces are simply not established. The second proposes that they are set up, but decay rapidly. This latter hypothesis would predict that amnesic patients would forget at a faster rate. A variant of this hypothesis is the proposal that one subgroup of amnesics—patients with damage to the hippocampus—forget faster than those with damage to other temporal lobe structures (Squire, 1981). In fact, given that the initial level of learning is equated, there is no evidence to suggest faster forgetting in either subgroup of amnesic patients (Huppert & Piercy, 1978a; Kopelman, 1985). Somewhat surprisingly, although any condition that compromises brain function is liable to lead to impaired learning, very few of these result in faster forgetting, once material has been acquired.

Retrieval

Warrington and Weiskrantz (1970) initially interpreted their puzzling results in terms of retrieval, arguing that the word-fragment cues that were so effective at test helped performance by ruling out potentially interfering responses. However, they subsequently rejected this interpretation on the grounds that other predicted effects did not occur (Warrington & Weiskrantz, 1978).

Contextual processing

An essential quality of episodic memory is that it allows individual specific memories to be retrieved. An influential theory as to what makes this possible proposes that individual episodes are linked to the specific time and place of the experience. This provides a way of specifying that experience, and subsequently retrieving that particular memory rather than others occurring at a different time or place. Loss of the capacity to link experiences to their spatial and temporal context would therefore grossly disrupt subsequent recollection. In a study using rats, Winocur and Mills (1970) observed that animals with hippocampal lesions were particularly bad at making use of environmental context in a spatial learning task, suggesting to Winocur (1978) that a failure to associate memories with context may also apply to human amnesic patients.

Evidence for a deficit in contextual memory came from an ingenious study by Huppert and Piercy (1978a, 1978b). They took advantage of the fact that people are very good at recognizing pictures that they have previously been shown, demonstrating first that the performance of amnesic and control patients can be roughly equated by giving the patients longer to encode the pictures than the controls. Their study involved presenting pictures either once or twice on each of 2 successive days. After the second day's presentation, participants were shown a sequence of pictures and asked to say whether they had seen each picture. If they recognized a picture they were then to decide on which day that picture had been shown.

The crucial comparison concerned the pictures that had been seen twice on day 1. Huppert and Piercy found that amnesic patients were more likely to say that items presented twice on day 1 had in fact been presented on day 2, presumably because the degree of familiarity was greater. The controls showed exactly the opposite pattern, being more accurate in assigning items to day 1 if they had been presented twice. Two presentations meant two chances of linking that picture to the day 1 context. In the absence of the link to context provided by episodic memory, the amnesic patients had to rely on a general feeling of familiarity. This did not allow them to distinguish between greater familiarity resulting from two presentations, and that resulting from a recent experience.

Schacter, Harbluk, and McLachlan (1984) showed a similar effect, using as their material the answers to Trivial Pursuit questions such as "What was Bob Hope's favorite food?", again finding that amnesic patients are bad at recalling *when* information is acquired tending to confuse recency with degree of familiarity. The term *source amnesia* has been applied to this characteristic difficulty that amnesic patients experience in recollecting the source of a given memory.

In Huppert and Piercy's (1978a; b) study, amnesic patients were more likely to say that pictures presented twice on day 1 had in fact been presented on day 2 demonstrating a deficit in contextual memory. © BSIP, MENDIL / Science Photo Library.

Amnesia: a modal model

In an attempt to pull together the overall pattern of data, I proposed what I termed a modal model of amnesia, a simple interpretation of the amnesic syndrome that appeared to capture most if not all of the evidence (Baddeley, 1998). This accepted a deliberately unspecified version of the consolidation hypothesis, whereby learning in episodic memory involved associating items with their context using some form of "mnemonic glue." This clearly nontechnical term was deliberately selected so as to indicate that it was *not* based on any sophisticated neurobiological evidence but simply accepted that a neurobiological interpretation of some form seemed necessary. This does not rule out a contextual hypothesis, as it could be argued that the essence of episodic memory is the capacity to "glue" experiences to a specific context, thus providing a contextual tag that allows individual experiences to be retrieved.

This simplified model of amnesia assumed that recall and recognition involved the same underlying storage processes, although they placed different constraints on subsequent retrieval. It assumed that semantic memory represented the residue of many episodes. Over time, the capacity to retrieve individual experiences might have been lost through forgetting, but it was assumed that those features that were shared by repeated episodes could be retrievable through a separate mechanism. Although this modal model seemed to give a plausible account of the classic amnesic syndrome, it was not clear how to test it and I myself ceased to work on amnesia.

Some years later, however, I was asked to talk about amnesia at a retirement symposium for Elizabeth Warrington. Because of our earlier work together, I agreed. I had not subsequently published anything on my speculative modal model of amnesia and thought it would be a good opportunity to obtain feedback from an expert audience. Despite absent-mindedly leaving my slides on the train en route, the talk seemed to go reasonably well. Then, shortly after the meeting, I was invited by Faraneh Vargha-Khadem, from the Institute of Child Health in London, to visit and test a patient, Jon, an experience that convinced me that my modal model of amnesia might well be wrong, or at any rate far too simple.

Developmental amnesia

Jon has the misfortune to have been born prematurely and had to spend his early days in an incubator. He suffered breathing problems resulting in anoxia, and substantial damage to his hippocampus. Somewhat unusually, this appears to be the limit of the damage to his brain. It was however, severe, with his hippocampus being less than half the normal size, and somewhat atypical in its structure.

At about the age of 5, Jon's parents began to suspect that he had memory problems, and this proved to be the case. However, despite having a degree of amnesia that made it difficult for him to cope independently, Jon has developed above average intelligence and good semantic memory skills. Furthermore, although his recall memory is clearly impaired, his recognition performance tends to be within the normal range. Figure 11.4 shows the performance of Jon and two matched control participants on the Doors and People Test (Baddeley, Vargha-Khadem, & Mishkin, 2001c). This test was developed to provide a separate measure for visual and verbal recall and recognition, allowing each of these components to be assessed separately and then combined to give overall visual scores, overall verbal scores, and combined

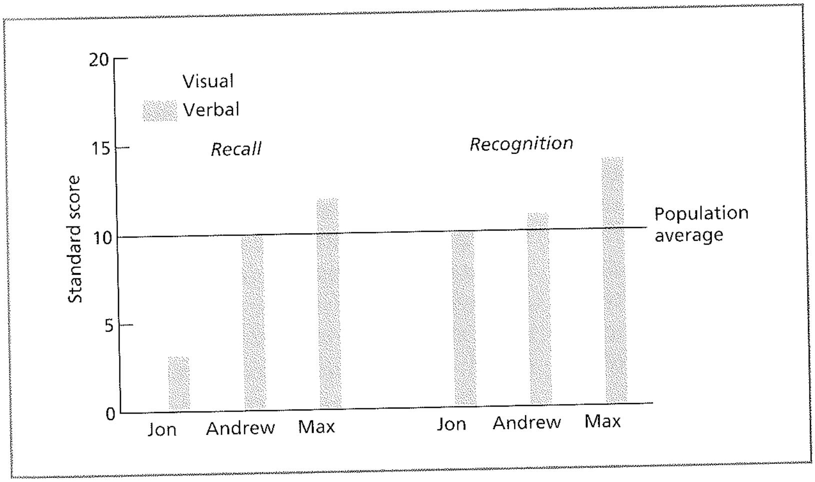


Figure 11.4 Performance on the Doors and People test of visual and verbal recall by Jon, a developmental amnesic patient, and two controls. Jon is impaired on recall but not recognition. From Baddeley et al. (2001). Copyright © 2001 MIT Press. Reproduced with permission.

recall versus recognition. A similar level of preserved performance was found on a wide range of other tests of recognition, confirming earlier observations on Jon and a number of other similar patients by Vargha-Khadem, Gadian, and Mishkin (2001), although subsequent studies suggest that Jon's recognition performance might not be completely normal (Gardiner, Brandt, Vargha-Khadem, Baddeley, & Mishkin, 2006).

Jon presents a number of problems for the proposed modal model of amnesia. First, if episodic memory is impaired how can semantic memory develop? Second, although the link between memory and intelligence is not clearly specified by the modal model, one might expect crystallized intelligence, based on prior learning, to be impaired, resulting, for example, in reduced vocabulary. This is not the case. Subsequent research suggests, however, that although Jon's knowledge of the world is excellent, it might take him longer than controls to acquire new facts (Gardiner, Brandt, Baddeley, Vargha-Khadem, & Mishkin, in press). Third, if recall and recognition involve essentially the same storage process, why, in Jon's case, can recognition be so well preserved and recall so impaired?

A hint as to the answer to this question comes from the previously described distinction between *remembering* based on the capacity to recollect a learning episode, to travel backwards in time to re-experience it, in Tulving's terminology, and the capacity to *know* that one has previously encountered an item, in the absence of recollective experience (see Chapter 1, p. 11). We attempted to assess Jon's capacity to "remember," but had considerable difficulty teaching him the distinction between remembering and knowing. Eventually, he declared that he understood the distinction and we went ahead. Jon used the remember-and-know categories about as often as controls. However, when control participants made a remember judgment they could describe their recollection, for example *The word "dog" reminded me of my granny's dachshund.* Jon did not. He reported that he tried to form a visual image of the cards on which the words had been presented, and if his image of the word was clear and bright then he categorized this as remembering. In short, he appeared to be using a strength rather than a recollective criterion.

Further evidence on this point came from an evoked electrophysiological response study that studied the electrical response of Jon's brain when attempting to recognize words that had previously been presented, and categorize them as "remembered" or "known." This study took advantage of the fact that "remember" judgments are accompanied by a specific activation pattern that differs from that evoked by "know" responses. Jon proved to lack the remember component (Düzel, Vargha-Khadem, Heinze, & Mishkin, 2001). Finally, Maguire, Vargha-Khadem, and Mishkin (2001) succeeded in finding a few memories from Jon's life that seemed to evoke the crucial episodic experience of reliving an event. When such memories were evoked in a brain-imaging study, he finally showed the standard "remember" pattern of brain activation, indicating that he does have the capacity to recollect but has great difficulty in laying down the memory traces on which such recollective experiences are based.

To summarize, it appears to be the case that Jon can learn, in the sense of building up familiarity, but that he is very impaired in his capacity to recollect. The fact that he has none-the-less acquired a rich semantic memory and above-average intelligence presents a clear challenge to theory and to earlier assumptions regarding the role of the hippocampus.

There is no doubt that Jon is far from typical as an amnesic patient. More specifically, Squire and colleagues have presented data from groups of amnesic patients who appear to have lesions limited to the hippocampus, and who behave in the standard way, with no evidence of preserved recognition memory (Reed & Squire, 1997; Manns & Squire, 1999). Why the difference? One possibility is that Jon acquired his hippocampal damage at a very early age whereas most amnesic patients become amnesic as adults. Given the greater potential plasticity of the young brain, it is possible that Jon's brain might have developed in such a way that the preserved tissue can supplement his damaged hippocampus. If this is the case, then one should find an association between the likelihood of finding a pattern such as that shown by Jon and the age of onset of the patient's amnesia. Testing this is not simple, as although similar cases to Jon are now being identified more widely, they tend not to have such a precisely limited area of neurological damage, or such a specific pattern of cognitive impairment. This issue is currently being pursued by Vargha-Khadem and colleagues, but the outcome is still unclear.

A second possibility is that the pattern of deficits shown by Jon reflects the specificity of the area of the damage within his brain, being limited to the hippocampus, whereas the surrounding regions comprising the *perirhinal* and *entorhinal* cortices appear to have been entirely spared. There is no doubt that some of the earlier claims for the importance of the hippocampus stem in part from associated deficits. In animal lesion studies, it is very difficult to lesion the hippocampus without influencing these areas, and in brain-damaged patients the damage is very rarely both extensive and confined to the hippocampus. In the case of the patients reported by Squire and colleagues (Reed & Squire, 1997; Manns & Squire, 1999), it is also possible that areas beyond the hippocampus might be compromised, in ways that are not readily detectable.

Aggleton and Brown (1999) proposed, prior to the study of Jon's memory, that whereas the hippocampus is important for episodic memory, familiarity-based recognition judgments might be based on the perirhinal regions that are preserved in Jon. They cite a number of cases from the literature with preserved recognition in the presence of clearly impaired recall, a pattern subsequently recorded by Mayes, Holdstock, Isaac, Hunkin, and Roberts (2002) in an adult-onset case in whom the sparing of recognition memory appeared to be associated with a lesion limited to the hippocampus.

So, does Jon really present a challenge to a simple modal model of amnesia, as I initially concluded? Or does he reflect the danger of drawing overgeneral conclusions from developmental cases? No doubt, as more cases appear and as our methods of assessing the extent of anatomical damage improve, this issue will in due course be resolved, as too will the role of episodic memory in the development of the semantic system.

RETROGRADE AMNESIA

Whereas anterograde amnesia refers to the incapacity to lay down new memories, retrograde amnesia refers to the impaired capacity to retrieve old memories. The two often go together, but are not highly correlated (Shimamura & Squire, 1991; Greene & Hodges, 1996; Kapur, 1999). For example, two patients studied by Baddeley and Wilson (1986) both had dense but pure amnesias with high and well-preserved intelligence, but one appeared to have excellent retrograde memory and could, for example, talk in great detail about his wartime experiences, whereas the other had at best only a hazy memory of his past. He knew he had been in the Navy and gone to university but could not remember in what order; he knew he had previously broken his arm, but could not recall how.

A few cases have been reported with the opposite pattern (Kapur, 1999). There is, however, often a possibility that the patient might be consciously or unconsciously avoiding recall of the period preceding the traumatic incident for emotional reasons (see Chapter 7 for further discussion).

Assessment of retrograde amnesia

Measuring the degree of retrograde amnesia presents one problem that is not shared with anterograde amnesia, namely that the tester typically does not have control over the learning of the material to be recalled, as learning might have occurred many years before. The first attempt to quantify degree of retrograde amnesia was made by Sanders and Warrington (1971), who presented their patients with photographs of people who were famous for a limited period at different points in time, finding that their amnesic patients typically performed more poorly on this task than the controls. They also observed that earlier memories were better preserved, so-called *Ribot's law*. This asserts that older memories are more durable than those acquired more recently (Ribot, 1882).

A number of similar scales have subsequently been developed using a range of material, including news events, winners of classic horse races, and TV shows that aired for a single season (e.g. Squire, Haist, & Shimamura, 1989). This general method suffers from two practical problems. First, the degree of knowledge of news events or races is likely to vary substantially across patients; second, scales of this sort are, of course, continually aging, as the recent events become progressively more remote, hence requiring a continuous process of revising and revalidating.

An alternative method is to probe the patient's memory of their earlier life by requesting autobiographical recollections, which can then if necessary be checked through a spouse or carer (see Chapter 7). Galton (1879) developed a cue word technique whereby a word is presented, for example, *mountain*, after which the participant is asked to recollect a personal event concerning a mountain, and then date the memory in time. Try it yourself, with the word *river*. What did you find?

In my own case, I recollected being in a rowing boat on a river, holding on to the bank and finding the boat drifting further and further out. Curiously, I can't remember whether I eventually fell in. I was about 16 at the time.

This probe method was further developed by Crovitz and Shiffman (1974) and used to study retrograde amnesia by Zola-Morgan, Cohen, and Squire (1983). Unfortunately, it is a somewhat laborious process; people with normal memory tend to produce large amounts of material, which must then be transcribed and evaluated, whereas amnesic patients who are principally of interest tend to produce much less.

In an attempt to reduce these methodological problems, Kopelman, Wilson, and Baddeley (1990) developed the Autobiographical Memory Interview (AMI), which involved asking people to remember specific information selected from a range of time periods. Some were remote, for example the name of their first school, others intermediate, such as their first job, whereas others probed more recent events, such as where the patient spent last Christmas. These were essentially factual questions that could be regarded as probing a form of **personal semantic memory**. In addition, for each life period, participants were asked to recollect a *specific* personal event. An example from childhood might be winning a race at school. These episodic recollections were then rated in terms of amount and specificity of information retrieved.

The test was validated using both healthy people and a range of patients and was found to be sensitive and reliable. Even patients with Korsakoff syndrome, who are commonly believed to be inclined to confabulate, produced either accurate recall, as validated by relatives, or simply said they could not remember (Kopelman et al., 1990). This and related scales have been used increasingly widely in line with the increased interest in autobiographical memory and its disorders (see Chapter 7). Retrograde amnesia generally leads to impairment in autobiographical memory on both the personal and the semantic scales. However, cases who show differential impairment do occur, some with personal memories intact (De Renzi, Liotti, & Nichelli, 1987) and others showing the opposite pattern (Hodges & McCarthy, 1993). This issue is discussed in more detail in Chapter 7 in terms of its implication for theories of autobiographical memory.

KEY TERM

Personal semantic memory: Factual knowledge about one's own past.

Kopelman et al.'s (1990) Autobiographical Memory Interview (AMI) measured the extent of retrograde amnesia by asking people to remember specific information selected from a range of time periods, and to recall a temporally specific personal event, such as winning a race. © H. Armstrong Roberts/Corbis.

Temporal retrograde amnesia gradients

When it occurs, retrograde amnesia tends to follow a somewhat different pattern across different patient groups. Patients with alcoholic Korsakoff syndrome show a steep decline in recent memories compared with patients who are alcoholic but without memory deficits (Zola-Morgan et al., 1983). A particularly interesting case is that of PZ, a distinguished experimental psychologist who had completed his memoirs shortly before succumbing to Korsakoff syndrome. Because of this, it was possible to identify events and facts that it was clear that he knew just prior to the onset of amnesia. As Figure 11.5 shows, he did indeed show a very striking loss of memory for people he had remembered well enough to describe in his autobiography, written shortly before the onset of amnesia.

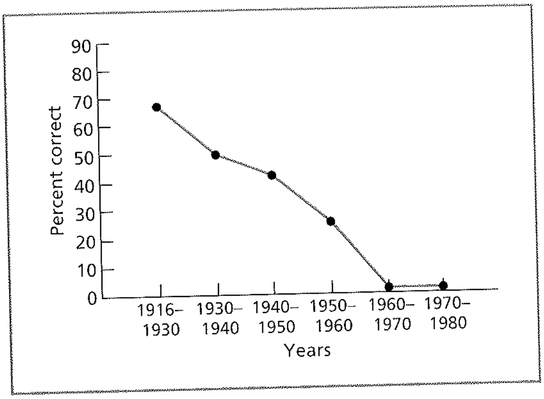


Figure 11.5 Patient PZ's retrograde amnesia gradient for information from his published autobiography. The earlier the information the better it is recalled. From Butters and Cermak (1986). Copyright © Cambridge University Press. Reproduced with permission.

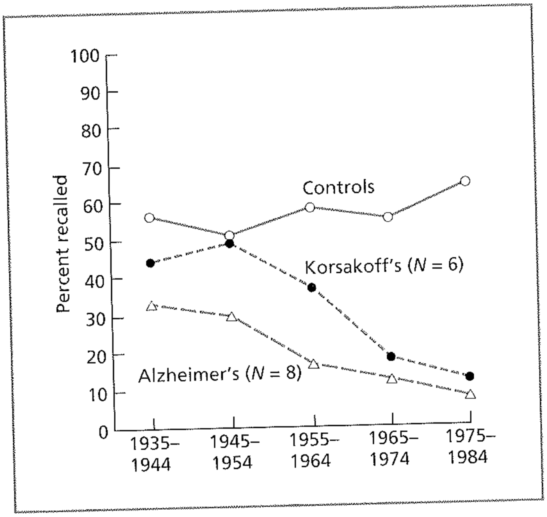


Figure 11.6 Percentage recall of famous news events as a function of when they occurred. Data from Kopelman (1989).

Kopelman (1989) compared the rate of decline in memory between two amnesic groups: patients with Korsakoff syndrome and patients suffering from Alzheimer's disease, and controls who were selected to be matched for age. As Figure 11.6 shows, the decline in memory for new events from different periods was much steeper in the Korsakoff patients. It is not clear why this should be the case. One might argue that the long periods of drinking by the Korsakoff patients might have resulted in fewer experiences worth remembering. However, it seems equally likely that the gradual onset of Alzheimer's disease would have had a similar effect. It seems more likely that the discrepancy reflects differences in the causes of the two amnesias at a neurobiological level.

Explanations of retrograde amnesia

There have been fewer studies of retrograde than anterograde amnesia, and less extensive theoretical analysis. This has begun to change in recent years with a number of models proposed, often accompanied by computer simulations to check that they are indeed able to predict the results claimed. Three of these models—those of Alvarez and Squire (1994); McClelland, McNaughton, and O'Reilly (1995); and Murre (1996)—differ in detail, but all assume that the hippocampus and surrounding regions play a crucial role in memory consolidation. Unlike the consolidation assumed to operate at the cellular and subcellular levels described in Chapter 4 (see p. 88), the explanation in this case refers to **system consolidation**, the process whereby information is consolidated within the brain by a process of transfer from one anatomically based system to another. These two types of consolidation are not, of course, mutually exclusive, although failure to consolidate at a cellular level will presumably interfere with any subsequent system consolidation.

The above three models differ in detail but all assume that the hippocampus and associated regions act as an intermediary, detecting and storing novel information at a relatively rapid rate, then holding it while it is gradually transferred to more cortical areas. Unlike hippocampal storage, which is relatively rapid but temporary, links within the cortex are assumed to take longer to set up, but are more durable. This consolidation process continues to progress within the neocortex after traces have been lost from the hippocampus, with the result that memory traces that have been in the brain for many years will be particularly robust, thus accounting for Ribot's law, the greater durability of early memory traces.

An alternative model is offered by the Multiple Trace Hypothesis, proposed by Nadel and Moscovitch (1997, 1998). They argue for the role of the hippocampus in retrieval, as well as encoding. They accept a version of the model just described, which they refer to as the "standard model" but assume that the process of long-term consolidation sets up recorded traces of experience within the hippocampal complex, rather than the neocortex. They assume that this will lead to multiple hippocampal replicas of earlier experiences. The temporal gradient in retrograde amnesia is assumed to result from partial damage to the hippocampus removing some of the available traces. Older traces, being more numerous, will be more likely to survive. However, complete damage to the hippocampus should lead to total retrograde amnesia. The question of whether this or one of the more standard models gives the better account of retrograde amnesia remains an open question (Sara, 2007).

In recent years, there has been an increasing interest in the general process responsible for memory consolidation. A study in which rats became familiar with a novel environment enabled Wilson and McNaughton (1994) to identify and monitor *place cells*, individual cells within the hippocampus that become activated when the rat approaches a particular part of the learned environment. They noted that during slow-wave sleep, the deepest sleep

KEY TERM

System consolidation: Process of gradual reorganization of the regions of the brain that support memory. Information is consolidated within the brain by a process of transfer from one anatomically based system to another.

level, such place cells were reactivated, as if some process of transfer or consolidation was taking place. More recently, the neural activity generated by daytime singing in birds has been found also to occur during sleep (Dave & Margoliash, 2000).

Studies in humans suggest that sleep deprivation can interfere with the process of consolidation. In one study, Stickgold, LaTanya, and Hobson (2000) showed first of all that a visual discrimination task led to maximum improvement several days after training, despite an absence of further practice. However, this impairment was not shown by a group who were deprived of a night's sleep immediately after learning. Evidence for the importance of sleep in word learning comes from a series of studies by Gaskell and Dumay (2003). They describe a technique whereby it is possible to detect whether a new word has been fully registered in the mental lexicon by the extent to which it is capable of interfering with the processing of existing words. They taught their participants word-like new items, e.g. *cathedruke*, establishing that it was in episodic memory by recall, and then testing the extent to which they slow down the processing of a close existing word such as *cathedral*. Recall is excellent immediately after learning but the new word does not interfere with the old. When tested after a night's sleep, clear interference is found, an effect that lasts for many months. It appears, therefore, that sleep is necessary for a new item to be consolidated into a person's verbal lexicon.

The tasks considered so far are implicit. However, a similar pattern of results was found by Gais, Albouy, Boly, Dang-Vu, Darsaud, Desseilles, et al. (2007) in a study involving presenting participants with 90 pairs of concrete nouns (e.g. *rabbit – chair*) and instructing them to form an image incorporating the two. Some 30 minutes later they were asked to recall as many pairs as possible, and then were kept awake for the next 24 hours. Forty-eight hours later, after two nights of full sleep, recall was again tested. A second group was treated in exactly the same way except that these participants were sleep deprived on the second night after having slept following their initial recall. Neuroimaging using fMRI during the 48-hour delayed recall test showed a different pattern across the two groups, with the group that had slept immediately after learning showing greater activation in the hippocampus and evidence of active links between the hippocampus and the medial prefrontal cortex, suggesting that these areas had benefited most from sleep-based consolidation.

The study of memory disorders resulting from damage to clearly specified areas of the brain has proved enormously useful in developing our theories of memory and our knowledge of brain function. Of great practical significance, however, are those conditions for which a serious memory deficit is a prominent but not inevitable symptom, and where the association between the deficit and its anatomical localization is often unclear. From the patient's viewpoint, however, regardless of its origin, a memory deficit can be a crippling affliction. It is therefore important to study memory performance in diseases of this type, and to try to develop methods of helping patients to cope with the associated memory problems. One such disease will be described next, namely traumatic brain injury; a second—Alzheimer's disease—is discussed in Chapter 13, which is concerned with aging.

TRAUMATIC BRAIN INJURY

Traumatic brain injury (**TBI**) occurs when the head receives a sharp blow, for example as a result of a fall, or is subject to a sudden

KEY TERM

Traumatic brain injury (TBI): Caused by a blow or jolt to the head, or by a penetrating head injury. Normal brain function is disrupted. Severity ranges from "mild" (brief change in mental status or consciousness) to "severe" (extended period of unconsciousness or amnesia after the injury).

acceleration or deceleration as in a car crash. The brain swirls around, resulting in damage from the bony protuberances within the skull, and from the twisting and shearing of fibers within the brain.

A few years ago I was waiting in a line of cars to leave a side road near a sea-coast resort when suddenly a blue figure arced in the air to the horror and consternation of onlookers. It was a motorcyclist hit by a car turning into the side road, resulting no doubt in one more serious head injury. Such injuries happen mainly to young men, and in the UK over 95% will survive with varying degrees of handicap. A major feature of this is difficulty in concentrating and remembering.

What sort of memory problems might our unfortunate motorcyclist expect? First of all, if the brain injury was severe, he might be expected to be in a coma, sometimes for many weeks. Indeed in the most serious cases the patient may be left in what is known as a *persistent vegetative state* in which physical functions continue to perform but mental functions do not. This in turn leads to the terrible ethical problem as to how long one should artificially maintain life in such a case. Fortunately, in most cases there will be a gradual recovery, often so gradual that it can be missed by the medical support staff. To optimize this process of monitoring, Shiel, Wilson, McLellan, and Watson (2000) developed a scale entitled the Wessex Head Injury Matrix Scale (WHIM), which picks up the tiny changes that occur in behavior as the brain slowly recovers from major trauma.

On recovering consciousness, the patient is likely to move into a state of *post-traumatic amnesia* (PTA), in which attention can be disturbed and the capacity for new learning grossly impaired. Once again, it is important to be able to monitor this gradual recovery, and to do so a number of scales have been devised (Levin & Hanten, 2002). A study by High, Levin, and Gary (1990) monitored the progress through PTA of 84 patients whose traumatic brain injury was sufficient to lead to coma. They typically first recovered *personal knowledge*, who they were; followed by *place*, where they were; and finally *temporal orientation*. The estimated date was typically displaced backwards, especially in more severe cases, where there could be an error of up to 5 years. As the patients recovered, the degree of error reduced, reflecting a shrinkage of retrograde amnesia.

Length of time in PTA can vary considerably, and provides a rough, although not infallible, guide to level of probable recover (Levin, O'Donnell, & Grossman 1979). Having recovered from PTA, the patient is likely to be left with a degree of retrograde amnesia. This might initially be quite extensive, but will shrink over time, as in the classic case described below.

> *A green-keeper, aged 22, was thrown from his motorcycle in August 1933. There was a bruise in the left frontal region and slight bleeding from the left ear but no fracture was seen on X-ray examination. A week after the accident he was able to converse sensibly and the nursing staff considered that he had fully recovered consciousness. When questioned, however, he said that the date was February 1922, and that he was a school boy. He had no recollection of 5 years spent in Australia and 2 years in the UK working on a golf course. Two weeks after the injury he remembered the 5 years spent in Australia and remembered returning to the UK; the past 2 years were, however, a complete blank as far as his memory was concerned. Three weeks after the injury, he returned to the village where he had been working for 2 years. Everything looked strange and he had no recollection of ever having been there before. He lost his way on more than one occasion. Still feeling a stranger to the district he returned to work; he was able to do his work satisfactorily but had difficulty in remembering what he had*

> *actually done during the day. About 10 weeks after the accident the events of the past 2 years were gradually recollected and finally he was able to remember everything up to within a few minutes of the accident. (Russell, 1959, pp. 69–70)*

The shrinkage in degree of retrograde amnesia is variable and typically less dramatic than that shown by our Australian green-keeper. The dense period of continuing amnesia immediately preceding the TBI is, however, very characteristic. Is the problem one of registering the experience in the first place, or consolidation of the memory trace? Light is thrown on this issue by a study by Yarnell and Lynch (1970) of American football players who have been "dinged." As they were led off, the investigator asked the name of the play that had led to the collision (e.g. Pop 22). Typically, the player could remember it immediately, but not when questioned later. Although other interpretations are possible, this certainly is consistent with a lack of early neural consolidation of the memory trace.

In Yarnell and Lynch's (1970) study of American football players the player could generally recall the name of the play that had led to the collision immediately, but not when questioned later.

There has, in recent years, been a growing interest in the long-term effects of playing high-contact games like American football and rugby league football. Gina Geffen, a neuropsychologist in Adelaide, Australia, was asked to examine an Australian-rules football player who had sustained a head injury. To obtain a comparison group, she tested a number of his colleagues using a test of speed of the semantic processing developed by Baddeley, Emslie, and Nimmo-Smith (1992). This involves the patient in reading a series of brief sentences that are either obviously true or obviously false. Typical positive sentences are *Nuns have religious beliefs* and *Shoes are sold in pairs*. Negative sentences are created by recombining positive instances, as in *Shoes have religious beliefs* and *Nuns are sold in pairs*. Go to Box 6.2 in Chapter 6 to try the test yourself.

Geffen found that not only her patient but also his team-mates in this extremely vigorous sport were somewhat impaired on this sensitive speed test of semantic processing (Hinton-Bayre, Geffen, & McFarland, 1997). Others have found similar results in other high-contact sports players, and regular testing has now become an important feature within American football. This residual deficit is of course much less severe than that found in PTA, and in American college football players appears to resolve within a few days (McCrea, Guskiewicz, Marshall, Barr, & Randolph, 2003).

What happens when patients recover from the temporary condition of PTA? Unfortunately, persistent problems of episodic memory tend to remain. Oddy, Coughlan, Tyerman, and Jenkins (1985) questioned patients and their carers some 7 years after a moderate or severe head injury. They found that 53% of the patients reported memory problems; this rose to 79% if one used the report of the carer as an index of impaired memory. Such a result raises three questions: Why do the two estimates differ? Which of them is more accurate? And how do they relate to actual performance on objective memory tests?

A probable answer to all three was provided by Sunderland, Harris, and Baddeley (1983).

We studied mild to moderate head-injured patients, some a few months and others a few years after their injury. Objective performance was measured using a number of tests that were known to be sensitive to TBI, including paired-associate learning, word and face recognition, performance on the previously described semantic processing test, and a test involving immediate and delayed recall of a short prose paragraph. In addition, patients and carers were both asked to complete a questionnaire regarding their memory problems, and in addition to keep a regular diary in which memory lapses were recorded.

As expected, both recent and more remote TBI groups performed more poorly than a control group on the various tests of memory and attention. This effect was equivalent across the two groups, regardless of whether the TBI was recent or more remote, suggesting that unfortunately the memory deficit tends to persist. The subjective measures also showed a difference between the ratings for patients and control participants. The central interest, however, was the relationship between the objective memory measures and memory complaints. This tended to be low for all except the prose passage recall, with correlations being particularly low for self-report by patients, and lower for questionnaires than diaries. We suggested that this was because the questionnaire measure itself depended on remembering, and that the patients simply tended to forget that they had forgotten. It is also the case that amnesic patients can be unaware of their memory deficit, even though it is very obvious to all around them. One densely amnesic patient I tested frequently expressed surprise at her continued failure to recall throughout a test session, remarking at frequent intervals that "I pride myself on my memory." Such lack of insight can make rehabilitation difficult, but fortunately does not occur in all amnesic patients.

The lack of correlation between most of the objective memory measures and reported lapses is worrying, given that such measures play an important role in assessing, advising, and helping the patient. However, this problem was already being tackled by Barbara Wilson, a clinical neuropsychologist working at Rivermead Rehabilitation Centre in Oxford.

Wilson came from a background of treating learning-disabled people using behavioral methods initially influenced by BF Skinner's approach to learning based on operant conditioning. This involves a very pragmatic approach based on closely monitoring the patient and systematically rewarding the relevant behavior. Wilson decided to develop a measure based on the capacity of patients to perform a range of practical memory tasks that were selected as being those on which she and carers had observed that patients were most likely to suffer memory problems in their everyday life. The resulting Rivermead Behavioural Memory Test (RBMT) comprised the tasks shown in Table 11.1.

We validated the RBMT using a large sample of patients who had suffered brain damage, from a range of different causes, correlating performance with reports of memory lapses by therapists who kept a record over many hours of treatment (Wilson, Cockburn, Baddeley, & Hiorns, 1989b). The correlations proved to be high and, as expected, the test proved superior to more standard measures of memory performance in predicting such everyday lapses.

Of course, the acid test for a measure that hopes to predict everyday function must depend on a correlation with performance in everyday life. Wilson (1991) was able to achieve this by following-up a total of 43 patients with severe memory disorders, all of whom she had tested some 5–10 years before. She took as her measure of coping the extent to which patients were able to live an independent life. The results are shown in Figure 11.7, together with the equivalent prediction based on a standard clinical assessment, the Wechsler Memory Scale (WMS). As can be seen, the RBMT does an excellent job, the two exceptions being cases that were not in fact amnesic, one having a phonological STM deficit and the other being a rare case of retrograde amnesia in the absence of anterograde. The WMS

TABLE 11.1: The components of the Rivermead Behavioural Memory Test (RBMT). The items were chosen to probe performance on aspects of everyday memory that were potentially problematic for patients.

The Rivermead Behavioural Memory Test	
1	Remembering a new name—first name.
2	Remembering a new name—second name.
3	Remembering a belonging.
4	Remembering an appointment.
5	Picture recognition.
6	Newspaper story—immediate and delayed recall.
7	Face recognition.
8	Remembering a new route—immediate recall.
9	Remembering a new route—delayed recall.
10	Remembering to deliver a message.
11	Orientation.
12	Date.

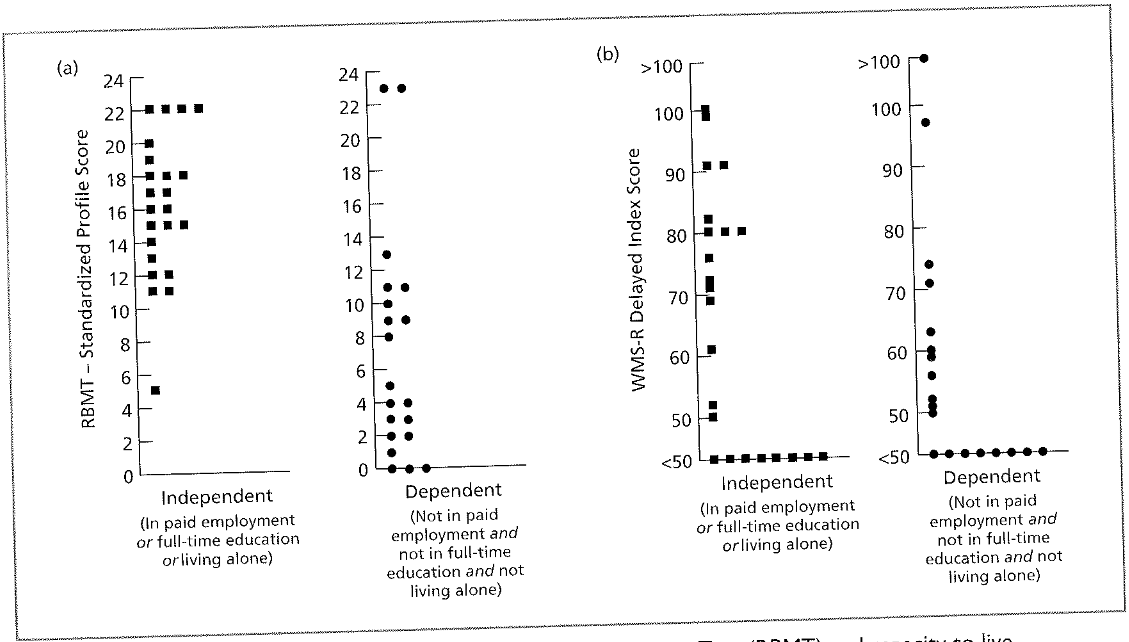


Figure 11.7 (a) Performance on the Rivermead Behavioural Memory Test (RBMT) and capacity to live independently. A follow-up of patients with memory problems. (b) Performance on the Wechsler Memory Scale—revised on the independent and dependent group. From Wilson (1991). Copyright © 1991 Psychology Press.

fairs rather less well. That does not, of course, indicate that it is a poor test; it is highly sensitive to memory impairment but was not designed to predict everyday problems. Both types of test are needed, together with theoretically based measures that can analyze the nature of the memory deficits observed more precisely than either of these, and potentially, by identifying the patient's strengths and weaknesses, facilitate subsequent treatment.

SUMMARY

The study of amnesia is driven by important clinical problems, and both benefits from, and enriches, theories of normal memory function. It is a very good example of the way in which theory and practice can fruitfully interact. This is shown in the classic amnesic syndrome in which a patient may have a gross deficit in episodic memory, but be otherwise cognitively unimpaired, with clear implications for the nature of long-term memory.

The amnesic syndrome typically involves damage to a circuit linking the temporal lobes, frontal lobes, and hippocampus. This can result from a number of diseases, including alcoholic Korsakoff syndrome, bilateral brain lesion, anoxia, or brain infection. Although the amnesia can be pure, in most cases the deficit will be accompanied by other cognitive problems.

It is important to distinguish between anterograde amnesia, reflecting the difficulty in acquiring new information, and retrograde amnesia, loss of access to memories from before the amnesic episode. Pure anterograde amnesia involves a substantial deficit in the acquisition of new memories, whether based on visual or verbal material, and whether tested by recall or recognition. In the amnesic syndrome, working memory is typically well preserved, as is access to semantic memory, although the capacity to add new information to semantic memory is typically defective. Implicit memory is also preserved, as measured by studies of priming, procedural learning, or classical conditioning. Rate of forgetting is typically unimpaired.

Early theories of amnesia in terms of levels of processing or proactive interference have now been superseded by variants of a contextual learning interpretation, which might reflect impaired consolidation. Most theories accept a role for the hippocampus, but surrounding areas are almost certainly also important. There is currently controversy as to whether the hippocampus is necessary for recognition memory, but general agreement that it plays a role in the subjective feeling of "remembering" that is sometimes regarded as essential to episodic memory.

Retrograde amnesia refers to the failure to access earlier memories. Methods of assessment include questionnaires requiring the recall or recognition of public events from different time periods, cueing autobiographical memories by Galton's single word probe method, and more structured autobiographical memory interviews.

These typically show a gradient of impairment, with earlier memories being better preserved, so-called Ribot's law. Theories of retrograde amnesia typically involve some version of the concept of consolidation, whereby information is transferred from the hippocampus to the cortex. Recent research suggests that sleep may play an important role in such transfer.

An important practical source of memory problems is that of traumatic brain injury (TBI), in which a blow or sudden acceleration or deceleration from a car crash causes disruption of brain tissue. A severe TBI will initially lead to coma, followed by a period of post-traumatic amnesia (PTA), a confused state in which new learning is impaired. PTA typically resolves gradually to leave a milder but more permanent memory deficit.

Rehabilitation of such patients requires adequate assessment. Tests of everyday memory play an important role in identifying problems and helping patients. Tests that are sensitive to memory deficit in general may be less good at predicting everyday memory problems. Specific tests of everyday memory have now been devised that successfully predict the severity of such difficulties, potentially important for rehabilitation. As such they provide a valuable addition to existing tests that focus on the issue of exactly what cognitive capacities have been impaired.

FURTHER READING

- **Baddeley, A. D., Kopelman, M. D., & Wilson, B. A.** (Eds.) (2002). *The handbook of memory disorders* (2nd edn). Chichester, UK: Wiley. A good source of evidence on specific types of memory disorder. It contains chapters on various types of memory deficit, and on ways of helping patients cope with memory problems.

- **Baxendale, S.** (2004). Memories aren't made of this: Amnesia at the movies. *British Medical Journal*, *329*, 1480–1483. An amusing analysis of the way in which amnesia is portrayed in movies, and a discussion of the implications of this for the public perception of memory and its deficits.

- **Parkin, A. J.** (Ed.) (1997). *Case studies in the neuropsychology of memory*. Hove, UK: Psychology Press. Accounts of individual patients that give a feeling for the way in which different forms of memory disorder influence the lives of patients.

- **Parkin, A. J., & Leng, N. R. C.** (1993). *Neuropsychology of the amnesic syndrome*. Hove, UK: Psychology Press. Although now somewhat dated, this presents a very clear account of the amnesic syndrome.

- **Wearing, D.** (2005). *Forever today*. New York: Doubleday. Deborah's Wearing's account of the devastating amnesia suffered by her husband, Clive, casting light on the human cost of severe memory disorder.